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*Mussel Poisoning**

By HERMAN SOMMER AND KARL F. MEYER,** San Francisco

The paralytic form of shellfish poisoning has been recognized as a distinct clinical entity for over a century.[1] It is to be distinguished from the allergic type, which affects only the people who are hypersensitive to the particular sea food, and it differs distinctly from the gastro-intestinal form, which is caused by spoilage of the mussels or pollution in the water. The symptoms of paralytic mussel poisoning are due solely to a neuropoison and consist of a tingling sensation in the extremities, followed by numbness, and in severe cases by complete paralysis and death within a few hours.

Although the malady has long been known, its cause is still veiled in darkness. One reason for this, undoubtedly, is the scarcity of its occurrence. In the European literature of the last one hundred years only about fifteen outbreaks have been recorded, with a total of some 120 cases and twenty-four deaths. Since all the mussels involved came from limited areas, such as estuaries or harbors, the assumption was natural that local factors, and probably land drainage, had something to do with the formation of the poison.

TABLE I. Epidemiology of Mussel Poisoning on the Pacific Coast, 1927-1934

Area	1927	1928	1929	1930	1931	1932	1933	1934	Total
Central California	102 (6)	------	*62(4)	2	------	**40(1)	7	------	213 (11)
Northern California and Oregon	------	------	------	------	------	------	15 (1)	------	15 (1)
Alaska	------	------	------	------	------	------	------	12 (2)	12 (2)
Totals	102 (6)	------	*62 (4)	2	------	**40(1)	22 (1)	12 (2)	240 (14)

*Including six (three) cases of clam poisoning.
**Including three cases of clam poisoning. Number of deaths in parentheses.

On the other hand, the recent outbreaks in California have afforded ample opportunities to gain a broader viewpoint and to add to the knowledge of this matter. The most extensive outbreak of mussel poisoning occurred near San Francisco in 1927, with 102 cases and six deaths. Others followed in 1929, 1932, 1933 and 1934, so that at present some 240 cases, with fourteen deaths, are on record. They all occurred between June and August, and resulted from mussels gathered between the Monterey peninsula and Fort Bragg, with a smaller outbreak in September, 1933, in the area between Crescent City, California, and Coos Bay, Oregon. Quite recently a report from Juneau, Alaska, indicates that several cases occurred, with two fatalities, in that neighborhood in July, 1934. All the mussels involved were freshly gathered along the open ocean shore, remote from stagnant or polluted waters, and were consumed fresh. The mussels having to do with one outbreak came from a coast line of over one hundred miles. These epidemiological records alone would demonstrate that any local factors or land drainage could not have been involved, but that mussel poisoning was more likely a marine phenomenon. (Table 1.)

These contentions were fully substantiated and considerably extended through laboratory examinations

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* From the George Williams Hooper Foundation, University of California, San Francisco.
Read at the Annual meeting of the American Public Health Association at Pasadena, September 3, 1934.
** Coworkers of the Experimental Part: R. Stohler, H. Mueller and W. F. Whedon.

started in 1927 and carried on to this date. One of the results is a poison curve for mussels from the vicinity of San Francisco, extending over a period of some seven years. As indicated in Figure 1, every year during the summer months the toxicity of the mussels rises to a peak, varying in intensity from year to year and making its reappearance some time between June and September. During some years the peak will reach the danger line; in others it will not. A comparison of the epidemiological data and the poison curve indicates a close correlation between the number of cases and the degree of toxicity of the mussels. This may be taken as a proof that the laboratory test, consisting of the intraperitoneal injection of a mussel extract in mice, gives an accurate measure of the toxicity of the mussels for humans. That the number of cases in the neighborhood of San Francisco has been steadily falling off is no sign of a decrease in toxicity, but indicates a more cautious attitude of the population in regard to the consumption of these shellfish. A second rise of lower intensity during the early spring months will be noticed. Furthermore, the decisive drop in toxicity to a minimum in late fall is noteworthy. At that time the poison may disappear entirely, or it may persist at a low level until the early spring of the following year. There are two phenomena, then, which need explanation: (a) the almost constant presence of minimal amounts of poison; (b) the strong increase in summer time. Both these findings are abnormal, since control mussels from La Jolla, San Diego, have never shown either of these characteristics. It is interesting to note, however, that poisonous mussels have been found not only along the coast from Monterey, California, to Coos Bay, Oregon, but also in samples received last year from Puget Sound, Washington.

That shellfish poisoning is, indeed, a marine phenomenon was proved by further laboratory studies, together with field observations. It was found that mussels gathered at the lowest possible level are generally more poisonous than those growing higher up on the rocks. This disproves the popular belief that exposure to the heat of the sun is responsible for the production of the poison. A comparison of the various kinds of clams points in the same direction. It should be recalled that in August, 1929, three persons died from the consumption of Washington clams (Saxidomus nuttallii), gathered north of San Francisco. Tests showed that, of the dozen varieties of edible clams in this area, the most dangerous are the Washington and Horseneck clams, or those which receive the largest amount of fresh ocean water; while oysters and soft-shell clams (Mya arenaria), or those which grow in the quiet bay waters at a distance from the open ocean, are entirely harmless. This fact strongly suggests that the poison factor makes its appearance with the water. That the poison itself also disappears with the water is proved by the fact that poisonous mussels kept in the laboratory give off their toxin into the water, and lose one-half of their toxicity in about ten days. This decrease in toxicity is also noticeable if the mussels are kept in a dry state, again refuting the argument that the harmful shellfish may have spoiled previous to consumption.

Attempts to demonstrate the poison itself in the water have long been unsuccessful. A better knowledge of its chemical characteristics finally helped to overcome the difficulty.[2] It was found that the poison may readily be removed from an aqueous solution by permutit. The fact that this synthetic silicate takes up only basic, organic as well as inorganic substances, proves definitely that the poison is of basic nature. This property was also of great help in the isolation of the substance from the water. Since the food of the mussel, the plankton, has long been under suspicion, large amounts of it were collected by filtration through plankton nets. The filter residue not only consists of plankton, but also of a large amount of microscopic sand, marine bacteria and detritus. Subsequently it was found that the sand contains a component which acts in a manner analogous to permutit, and which will take up the poison whenever it is in contact with its aqueous solution. It was natural, then, that the small amount of poison finally demonstrated in the water, i.e., about a lethal mouse dose per bucket, was found adsorbed on the sand. Where it originally came from is still a matter of conjecture; it may have been contained in the plankton, or it may have been excreted by near-by mussels. What practical importance the floating sand may have as a carrier of the poison remains for further investigations.

Another possible approach to the study of the plankton in relation to the poison is, of course, the statistical method. Plankton counts, especially a detailed classification of all dinoflagellates, carried on for nearly three years, tend to show that several species of the genus Gonyaulax occur most frequently, and often in large numbers, just before and during the poison seasons. Besides, a physiological change in these organisms seems to occur frequently at about the same time, manifesting itself by a more intense pigmentation. While the ratio of the number of Gonyaulax to the degree of toxicity is by no means constant, it is not unlikely that a detailed study of these and other peridinials occurring at times in large numbers might yield interesting results.

In this connection it should be recorded that during the poison period mussels are usually found with full

intestinal tracts and enlarged digestive glands. This is of frequent, but by no means constant occurrence. In fact, there is not one characteristic which may be used as an absolutely sure distinguishing feature between normal and harmful bivalves. The general reactivity of the mussels, their respiratory quotient, the histological picture, the bacterial flora and protozoan fauna are some of the characteristics which have been studied and found more or less identical in the two groups of mussels. Unusual appearances along the ocean shore, like phosphorescence or "red water," have often been observed during the toxic season, but they can not be relied upon as warning signals. The poison time falls in the summer months, when marine life is most intense and unusual phenomena are apt to make their appearance.

One of the unusual occurrences which was noticed for several years during the peak of the poison curve was the pronounced mortality of sand crabs. Subsequent investigation proved that these crustaceans are very nearly of the same toxicity as the mussels of the same locality at the same time.[3] They may be used, therefore, as test animals when mussels are not readily available. Although both the bivalves and the sand crabs are plankton feeders, the latter show some significant differences in their response to the substance. Sand crabs, with full intestinal canals, are as much as ten times more poisonous than those with empty digestive organs, indicating a rapid excretion of the poison. Furthermore, the crabs seem to be affected by some pathological condition which manifests itself in brown, melanin-like spots, around the joints of their legs. A detailed investigation of these conditions should be expected to help considerably in the elucidation of the problem.

With the help of the sand crabs, the widely held notion that the spawning of the shellfish is in any way involved in this problem may be readily disproved. The poison periods for the crabs and the mussels coincide, while their spawning seasons vary considerably. The sand crabs spawn mostly during the summer, while the mussels deposit their sex products in greatest numbers during the fall months, when the toxicity has subsided.

It is strange that mollusks of such an intense toxicity that half a dozen of them are sufficient to kill a grown person should not show any outward signs of their poison. The explanation is that they contain a very small amount of an extremely toxic substance. Chemical studies have proved this. The purest preparations of poison so far obtained are lethal to mice in doses approaching one-millionth of a gram, on intraperitoneal injection. It is more active than any known chemical poison. Only the antigenic toxins of some plants and bacteria are more potent. In its action it resembles some of the most potent alkaloids. It acts quickly or not at all, and is rapidly excreted. It is not destroyed by boiling and not counteracted by any known drug.

Guarding against the poisoning is rather difficult. The best preventive method is education of the people in the collection and consumption of shellfish. The old saying that shellfish should be avoided during the months without "r" is well founded, and September and October ought to be added also. Since practically all the poison is concentrated in the digestive gland of the shellfish, this organ should be strictly avoided. The broth, which naturally contains a good deal of the harmful substance, should also be discarded, at least during the summer months. It is doubtful whether any accidents would occur, even during the poison season, if only the light meat were consumed. A German investigator in 1885,[4] and recently a coworker in this laboratory,[5] have proposed that mussels be boiled for twenty minutes with the addition of sodium bicarbonate, which destroys most of the poison. This method is to be highly recommended from a chemical standpoint, less so from a culinary point of view. In California a seasonal quarantine on the sale of mussels, based on the present studies, has been found most expedient and undoubtedly has helped greatly in keeping the number of poison cases down to a minimum.

Mussel poisoning in itself would hardly warrant the time and energy spent in investigation of this sort were it not that a number of marine poisonings may be closely related to this problem in one or another of its aspects. They all have in common one characteristic: they are capricious in their appearance, and elusive. Fugu poisoning is caused principally in Japan by the consumption of the liver and sex products of Tetroden fish. It resembles mussel poisoning in that the active substance, although a sex poison, can hardly be distinguished chemically or toxicologically from the mussel toxin. The fish poison of the subtropical American waters, Ciguatera, is also of rare and elusive occurrence. The Haff disease, limited to a small number of localities of northern Germany, again made its appearance, after a lapse of several years, during 1933. It also occurs during the summer months exclusively and is caused by the consumption of eel and similar fish from the brackish waters of the Haff lagoons near Koenigsberg. Whether the explanation given at present by the German workers is correct, namely, that the sewage from the lumber and paper mills is to blame, remains to be seen. Last, but not least, the water-bloom poisoning, which has again been reported in recent years, especially from the lakes of

Minnesota, must be mentioned, although it is of fresh-water origin. Cattle and other domestic animals may die in less than an hour after drinking from lakes which in summer are covered with the growth of blue-green algae. Here again an exceedingly potent substance, which is apparently liberated by some microorganism in the water, is to blame. Fitch, Gortner, and co-workers[6] have recently been able to demonstrate the poison in solution in the water.

Although the results of these studies are very incomplete, so far as the origin of this strange poisoning is concerned, the following conclusions seem justified. Mussel poisoning is definitely of marine origin. It has occurred for centuries, in the waters of the northern temperate zone, and it will undoubtedly occur again. There is nothing that can be done about it other than to study its peculiarities and guard against its effects. It is of rare occurrence, and experiences gained during the life of one generation have usually been forgotten by the next. It is hoped that the results of these studies will have thrown at least a little light on this interesting field of marine toxicology. —From *California and Western Medicine.*

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MORBIDITY

Complete Reports for Following Diseases for Week Ending August 24, 1935

Chickenpox
70 cases: Berkeley 3, Oakland 8, Antioch 1, Compton 1, Long Beach 3, Los Angeles 7, Montebello 1, Pasadena 3, Redondo 1, Santa Monica 1, Lynwood 1, Hawthorne 1, South Gate 1, Monterey County 1, Napa County 2, Orange County 1, Santa Ana 1, San Francisco 16, Stockton 5, Santa Barbara 10, Palo Alto 2.

Diphtheria
26 cases: Alameda County 3, Berkeley 1, Oakland 2, San Leandro 1, Imperial County 2, Glendale 1, Los Angeles 10, Pasadena 1, Sacramento 2, San Bernardino 1, Santa Barbara 1, Yuba County 1.

German Measles
36 cases: Alameda County 2, Albany 1, Berkeley 1, Hayward 1, Oakland 6, Contra Costa County 1, Los Angeles County 3, Compton 1, Long Beach 3, Los Angeles 3, Pasadena 3, Tustin 1, San Francisco 9, Stockton 1.

Influenza
10 cases: Berkeley 1, Los Angeles County 2, Los Angeles 6, San Diego 1.

Malaria
6 cases: Los Angeles 1, Riverside 1, Santa Paula 1, Yolo County 1, Yuba County 2.

Measles
89 cases: Alameda 2, Berkeley 2, Oakland 7, Angels Camp 1, El Dorado County 1, Los Angeles County 3, Burbank 1, Long Beach 1, Los Angeles 13, Monterey County 2, Orange County 1, Santa Ana 4, Sacramento County 1, San Bernardino 1, San Francisco 31, Lompoc 1, Santa Barbara 3, Santa Clara County 1, Palo Alto 1, San Jose 5, Santa Clara 2, Tulare County 1, Ventura County 1, Oxnard 1, Yolo County 1, California 1.*

Mumps
67 cases: Alameda 1, Berkeley 6, Oakland 13, Fresno 1, Beverly Hills 1, Long Beach 4, Los Angeles 9, Pasadena 1, San Marino 2, Santa Monica 1, Madera 1, Santa Ana 1, Laguna Beach 1, Sacramento County 3, Sacramento 5, San Diego 3, San Francisco 1, San Joaquin County 1, Stockton 1, Santa Maria 1, Siskiyou County 1, Solano County 1, Sonora 1, Yolo County 4, Davis 1, Woodland 2.

Pneumonia (Lobar)
26 cases: Oakland 5, Los Angeles 11, Montebello 1, Monterey Park 1, Monterey County 1, Sacramento County 1, San Diego County 2, San Diego 2, San Francisco 1, Santa Barbara 1.

Scarlet Fever
81 cases: Albany 1, Oakland 3, Colusa County 1, Fresno County 5, Fresno 2, Hanford 1, Los Angeles County 4, Alhambra 3, Burbank 1, Long Beach 1, Los Angeles 12, Pasadena 2, Santa Monica 1, Lynwood 2, Napa County 1, Anaheim 1, Huntington Beach 2, Riverside 3, Sacramento 4, Ontario 1, San Bernardino 1, Coronado 1, San Diego 2, San Francisco 2, San Joaquin County 9, Stockton 3, Tracy 1, Paso Robles 2, Burlingame 1, San Mateo 2, Santa Barbara County 1, San Jose 1, Vallejo 1, Healdsburg 1, Winters 1, California 1.*

Smallpox
2 cases: Santa Clara County.

Typhoid Fever
11 cases: Oakland 2, Contra Costa County 1, Fresno County 3, Los Angeles 3, Santa Clara County 1, Tulare County 1.

Whooping Cough
125 cases: Alameda 1, Albany 2, Berkeley 3, Oakland 18, Fresno County 4, Reedley 4, Inyo County 2, Los Angeles County 5, Alhambra 1, Glendale 2, Huntington Park 1, Inglewood 3, Long Beach 7, Los Angeles 17, San Marino 1, Monterey Park 2, Monterey County 2, Placer County 8, San Diego County 1, La Mesa 1, San Diego 14, San Francisco 13, San Joaquin County 1, Stockton 4, San Mateo County 1, Burlingame 3, Santa Barbara 3, San Jose 1.

Meningitis (Epidemic)
4 cases: Los Angeles 2, Lynwood 1, Santa Clara County 1.

Dysentery (Amoebic)
2 cases: Santa Barbara County 1, San Jose 1.

Dysentery (Bacillary)
2 cases: Los Angeles 1, Sacramento 1.

Pellagra
5 cases: Oakland 1, Los Angeles 2, San Francisco 1, Santa Barbara County 1.

Poliomyelitis
34 cases: Berkeley 1, Fresno County 2, El Monte 1, Glendale 1, Los Angeles 4, Sacramento County 1, Sacramento 1, San Juan Bautista 1, San Luis Obispo 1, Tulare County 9, Lindsay 1, Visalia 11.

Tetanus
One case: Los Angeles County.

Trachoma
3 cases: Los Angeles 1, Riverside County 2.

Encephalitis (Epidemic)
One case: Oakland.

Paratyphoid Fever
2 cases: San Francisco 1, San Joaquin County 1.

Trichinosis
One case: Kern County.

Food Poisoning
5 cases: Los Angeles County 3, Montebello 1, San Francisco 1.

Undulant Fever
2 cases: Los Angeles.

Tularemia
2 cases: Susanville 1, Ventura County 1.

Septic Sore Throat (Epidemic)
2 cases: Berkeley 1, Lompoc 1.

Relapsing Fever
2 cases: El Dorado County 1, San Bernardino County 1.

Rabies (Animal)
10 cases: Los Angeles County 1, Alhambra 1, Los Angeles 7, San Diego 1.

* Cases charged to "California" represent patients ill before entering the State or those who contracted their illness traveling about the State throughout the incubation period of the disease. These cases are no chargeable to any one locality.

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